WHAT IS CLAIMED IS:

Claim 1. A method for revascularizing an ischemic region, comprising the steps of:

(a) preparing a pharmaceutical composition comprising a recombinant fibroblast growth factor-1 (FGF-1); and

(b) injecting an amount of said pharmaceutical composition into the ischemic region, said amount being sufficient to induce local neoangiogenesis, wherein the FGF-1 is prepared by a process comprising the steps of:

(i) transforming an *E. coli* host cell with a plasmid comprising an expressible gene encoding a biologically active human acidic fibroblast growth factor protein, operably linked to a promoter;

(ii) infecting the transformed bacterial host cell with a bacteriophage λ which mediates delayed lysis; and

(iii) cultivating the *E. coli* host cell under a culture condition that induces lytic growth of said cell without lysis until a desired level of production of said protein is reached, wherein said protein is produced as a soluble, biologically-active human acidic fibroblast growth factor protein.

Claim 2. The method of claim 1, wherein the expressible gene has a sequence which is contained within the group consisting of SEQ ID NO: 1, SEQ ID NO: 3, SEQ ID NO: 4, and SEQ ID NO: 6.

Claim 3. The method of claim 1, wherein the bacteriophage λ has a temperature-sensitive mutation which is cI_{857}.

Claim 4. The method of claim 1, wherein prior to the cultivating step, the *E. coli* host cells are grown at a temperature between 20 to 37 °C which prevents lytic growth of the bacteriophage λ.

Claim 5. The method of claim 1, wherein the bacteriophage λ has a mutation in at least one gene which mediates delayed lysis.

Claim 6. The method of claim 5, wherein the at least one gene which mediates delayed lysis is selected from the group consisting of N, Q and R.

Claim 7. The method of claim 1, wherein the *E. coli* host cell produces a suppressor for the repair of amber-mutations.

Claim 8. The method of claim 1, wherein the *E. coli* host cell lacks a suppressor for the repair of amber-mutations.

Claim 9. The method of claim 1, wherein the infecting bacteriophage λ is provided at a multiplicity of infection in a range of about 1 to about 100.

Claim 10. The method of claim 1, wherein the infecting bacteriophage λ is provided at a multiplicity of infection in a range of about 10 to about 25.

Claim 11. The method of claim 1, wherein bacteriophage-mediated delayed lysis of the *E. coli* host cell is delayed at higher multiplicities of infection relative to lower multiplicities of infection.

Claim 12. The method of claim 1, wherein the human acidic fibroblast growth factor protein has the sequence as set forth in SEQ ID NO: 7.

Claim 13. The method of claim 1, wherein the promoter is a T7 polymerase promoter and the *E. coli* host cell comprises a gene for T7 RNA polymerase.

Claim 14. The method of claim 13, wherein the gene for T7 RNA polymerase gene is under the control of an inducible promoter.

Claim 15. The method of claim 14, wherein the inducible promoter is a lac UV 5 promoter.

Claim 16. The method of claim 1, wherein the biologically active human acidic fibroblast growth factor protein contains amino acids 9-155 as shown in SEQ ID NO: 2.

Claim 17. The method of claim 1, wherein the biologically active human acidic fibroblast growth factor protein contains amino acids 2-141 as shown in SEQ ID NO: 7.

Claim 18. The method of claim 1, wherein the biologically active human acidic fibroblast growth factor protein contains amino acids 2-135 as shown in SEQ ID NO: 5.

Claim 19. The method of claim 1, wherein the biologically active human acidic fibroblast growth factor protein comprises a sequence shown in SEQ ID NO: 8.

Claim 20. The method of Claim 1, wherein said FGF-1 is injected at a final concentration in a range of about 0.1 μg/kg body weight per site to about 10 mg/kg body weight per site.

Claim 21. The method of Claim 1 wherein said FGF-1 is injected at a final concentration in a range of about 10 to 100 μg/kg body weight per site.

Claim 22. The method of claim 1, wherein the pharmaceutical composition further comprises a physiologic glue.

Claim 23. The method of Claim 22, wherein said physiologic glue is fibrin glue.

Claim 24. The method of Claim 1, wherein said FGF-1 and said physiologic glue are mixed immediately prior to application.

Claim 25. The method of Claim 1, wherein said pharmaceutical composition further comprises an anticoagulant.

Claim 26. The method Claim 25, wherein said anticoagulant is heparin.

Claim 27. The method of Claim 26, wherein the heparin is applied at a final concentration in a range of about 1 U per ml to about 1000 U per ml.

Claim 28. The method of Claim 1, wherein said injecting step further comprises:

making a thoracotomy incision;

identifying the at least one site of coronary artery stenosis;

administering a β-blocker to reduce the heart rate to a range of about 20-60 beats per minute; and

injecting the pharmaceutical composition intramyocardially at or near the at least one site of coronary artery stenosis.

Claim 29. The method of Claim 28, wherein said thoracotomy incision further comprises an anterior left-sided incision; dissecting a region of costal cartilage over a 5^{th} rib; and opening a left pleural space and a pericardium.

Claim 30. The method of Claim 28, wherein the step of identifying the at least one site of coronary artery stenosis further comprises retracting the heart forward using traction sutures.

Claim 31. The method of claim 1, wherein the neoangiogenesis is long term and occurs in the ischemic region at 6 weeks after the injection.

Claim 32. The method of claim 1, wherein the neoangiogenesis is long term and occurs in the ischemic region at 3 months after the injection.

Claim 33. The method of claim 1, wherein the method further comprises performing a coronary artery bypass graft.

Claim 34. The method of claim 1, further comprising the step of injecting a composition comprising a physiologic glue subsequent to injection with the pharmaceutical composition.

Claim 35. A method for treating coronary artery disease in a patient, comprising the steps of:

(a) preparing a pharmaceutical composition comprising a recombinant fibroblast growth factor-1 (FGF-1);

(b) injecting an amount of said pharmaceutical composition into at least one site in a heart wall, said amount being sufficient to improve myocardial perfusion; and

(c) injecting a composition comprising a physiological glue to a surface of the heart at the site(s) where the pharmaceutical composition was injected, wherein the FGF-1 is prepared by a process comprising the steps of:

(i) transforming an *E. coli* host cell with a plasmid comprising an expressible gene encoding a biologically active human acidic fibroblast growth factor protein, operably linked to a promoter;

(ii) infecting the transformed bacterial host cell with a bacteriophage λ which mediates delayed lysis; and

(iii) cultivating the *E. coli* host cell under a culture condition that induces lytic growth of said cell without lysis until a desired level of production of said protein is reached, wherein said protein is produced as a soluble, biologically-active human acidic fibroblast growth factor protein.